without systolic dysfunction (n = 20). Before feeding 8% NaCl (N) and at compensated LV hypertrophic (Com) and heart failure stages (HF), LV diameter, LV pressure and LV tissue hydroxyproline content were measured. The amounts of mRNA for prepro endothelin-1 (ppET-1) and angiotensin converting enzyme (ACE) were determined by quantitative RT-PCR in endocardial and epicardial regions of LV.

Results: Following is characteristics of this model.

	N	Com	HF
LVEDP (mmHg)	6 ± 1	7 ± 1	20 ± 2*†
Fractional shortening (%)	43 ± 1	45 ± 2	39 ± 1
LV hydroxyproline (μmol/g)	3.3 ± 0.2	4.1 ± 0.3	5.6 ± 0.6*†

(mean ± SE, *p < 0.01 vs N, †p < 0.01 vs Com)

LVEDP increased at HF with an increase in LV hydroxyproline content in the absence of significant changes in fractional shortening. A microscopic study showed that interstitial fibrosis was predominant in the endocardial region rather than the epicardial region at HF. In the endocardial region ppET-1 mRNA significantly increased by 2.3-fold as compared with N but did not in the epicardial region. In contrast, ACE mRNA increased by 1.6-fold and 1.8-fold in the endocardial and epicardial regions, respectively.

Conclusion: Endothelin pathway and renin-angiotensin system may play different roles as autocrine/paracrine factors in the progressive LV remodeling in hypertensive hearts resulting in diastolic heart failure.

1235-73 Correlation Between Left Ventricular Mass, Carotid or Femoral Wall Mass, and Blood Pressure in a Non-selected Population

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Background: hypertrophy has proved to be an adaptive mechanism reducing mechanical stress both in LV and conduit arteries. A relationship between LV mass and arterial mass should therefore exist.

Methods: 503 randomly selected subjects (245 men) were investigated with echocardiocardiography and high-resolution ultrasonography of their carotid and femoral arteries. Left ventricular mass (LVM) was calculated from M-Mode values and indexed to body surface area. Arterial wall mass was calculated from intima-media wall thickness and internal diameter for both carotid (CM) and both femoral arteries (FM). BP were measured according to the WHO guidelines.

Results: The following table shows mean values and correlation coefficients for the aforementioned variables ($p < 0.001$ for all). The correlation coefficients between LV mass and arterial masses remained statistically significant after adjustment for age and sex ($p < 0.005$).

Variable units	SBP (mmHg)	DBP (mmHg)	LVM (gr·m^{-2})	CM (mg/mm)	FM (mg/mm)	Age (years)
means ± SD	*135 ± 28*	*88 ± 15*	*104 ± 24*	*9.5 ± 2.5*	*8.3 ± 2.5*	*44 ± 11*
LVM	0.44	0.40	-	0.38	0.53	0.19
CM	0.44	0.31	0.38	-	0.52	0.59
FM	0.45	0.40	0.53	0.52	-	0.58

Conclusions: There is a clear association between LVM and wall mass of carotid or femoral arteries which is not explained by differences in age, sex or body size but is mainly related to systolic BP values. Non-invasive arterial mass measurement allows to precise target organ status in hypertension. As already shown with LVM determination, this might be helpful for cardiovascular risk prediction.

1235-74 Normal Left Ventricular Ejection Fraction Is Associated With Midwall Dysfunction in the Presence of Concentric Left Ventricular Geometry

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Background: LV endocardial shortening (eS) is often supranormal in arterial hypertension; midwall shortening (mS) is rarely above the normal range and is often reduced, especially in the presence of LV concentric geometry.

Methods: M-mode eS and ejection fraction (EF, by z-derived method), were studied in 116 hypertensive patients with LV concentric geometry (relative wall thickness >0.44), 41 with normal (55 ± 9 years, 21 women) and 75 with depressed mS (54 ± 9 years, 21 women) and compared to a reference adult population of 537 subjects (46 ± 12 years, 224 women).

Results: Body mass index was identically higher in the patients' groups than in controls (both $p < 0.01$. Diastolic blood pressure was higher in patients with low than in those with normal mS ($p < 0.05$. Table shows the between-group comparison of LV mass and chamber function.

	Controls (n = 537)	Normal mS (n = 41)	Low mS (n = 75)
LV Mass Index (g/m$^{2.7}$)	34.7 ± 8.0	46.3 ± 13.7†	56.8 ± 16.1*†
Relative wall thickness	0.34 ± 0.05	0.47 ± 0.02†	0.54 ± 0.08*†
Ejection Fraction (%)	66.3 ± 4.9	71.4 ± 5.0†	64.3 ± 9.2
Endocardial Shortening (%)	36.4 ± 4.7	41.5 ± 5.1†	34.9 ± 8.4
LV Dimension (cm)	4.9 ± 0.5	4.6 ± 0.4†	4.6 ± 0.6†
Cardiac Output (l/min)	4.9 ± 1.1	4.8 ± 1.1	4.4 ± 1.1*†
Peripheral Resistance	1539 ± 356	1978 ± 481†	2277 ± 536*†

† $p < 0.05$ vs controls; * $p < 0.05$ vs normal mS

Conclusions: "Normal" LV chamber function in the presence of concentric geometry is associated with depressed midwall performance, more severe LV hypertrophy, lower cardiac output, higher peripheral resistance. Ejection fraction is not representative of the true myocardial function when LV geometry is altered.

1235-75 Left Ventricular Diastolic Function in Hypertensive Patients With ecg Determined Left Ventricular Hypertrophy: The LIFE Study

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Background: It has been shown that abnormal diastolic left ventricular (LV) function assessed by pulsed Doppler of mitral inflow velocities can be early markers of cardiac involvement in hypertensives. Whether this finding is related to LV hypertrophy (LVH), and if so, to a specific geometric pattern of LVH is currently unknown.

Methods: Echocardiograms were obtained recording 2-D, M-mode and pulsed wave Doppler of the mitral inflow velocity and the isovolumetric relaxation time (IVRT) in 488 patients with mild to moderate hypertension and LVH determined by ECG (Cornell voltage duration criteria ≥2,440 mm × msec or modified Solokow Lyon: SV1 + RV5/RV6 >38 mm) after 14 days of placebo treatment. IVRT is consistent with impaired relaxation and IVRT >100 msec is regarded as pathological in all ages.

Results:

	Normal	Concentric remodelling	Eccentric hypertrophy	Concentric hypertrophy	P
Number	92 (19%)	65 (13)	175 (36)	156 (32)	NS
E-peak (cm/sec)	65.7 ± 19	64.0 ± 18	67.0 ± 19	64.2 ± 17	NS
A-peak (cm/sec)	77.5 ± 20	78.1 ± 19	78.3 ± 21	82.8 ± 21	NS
E/A-ratio	0.88 ± 0.31	0.85 ± 0.27	0.91 ± 0.40	0.82 ± 0.32	NS
Deceleration time	216 ± 65	236 ± 67	222 ± 74	233 ± 67	NS
IVRT (msec)	110 ± 23	119 ± 24	118 ± 30	118 ± 30	NS
IVRT >100 msec	63 (68%)	49 (76%)	124 (73%)	116 (73%)	NS

Conclusion: Abnormal IVRT is highly prevalent in all LV geometric subgroups among hypertensive patients with ECG LVH. No difference in IVRT were other diastolic filling parameters was statistical significant among patient groups defined by LV geometric patterns.

1235-76 Development of Two Different Models of Hypertensive Heart Failure in Rats – Systolic Failure and Isolated Diastolic Failure

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Background: Adaptive LVH to hypertension is followed by a state of impaired LV diastolic and/or systolic function, ultimately by the transition to overt heart failure. Two different types of heart failure are observed in human hypertension: isolated diastolic failure (DF) with concentric LVH and preserved systolic function, and systolic failure (SF) with eccentric LVH and impaired LV contraction. There are some animal models which show SF, however models of isolated DF have not been established yet.

Methods: Dahl salt sensitive rats were placed on a 8% NaCl diet from various week-old to make different physiology. LV geometry and function were assessed by echo indexes and by pressure data.

Results: The model of isolated DF with concentric LVH and preserved LV fractional shortening (FS) was developed at around 19 weeks old by NaCl diet from 7 weeks old (n = 15). On the other hand, model of SF with eccentric LVH and impaired contraction was developed at around 26 weeks old by NaCl diet from 8 weeks old (n = 9). The rats in both groups showed the symptoms of heart failure reflecting elevated LVEDP. There were no significant differences in LVEDP between the two groups; however, peak + dP/dt was lower in the SF than in the DF.

Conclusions: Two different models of hypertensive heart failure, isolated diastolic failure and systolic failure, were newly developed in Dahl rats. These